AMENDMENTS TO THE SPECIFICATION

Please amend the paragraph [0244] at page 70 of the specification as shown below:

Affinity chromatography using glutathione *S*-transferase (GST) fusion proteins confirmed that the cloned interacting protein bound specifically to the DOR tail and not to the GST that lacked the tail sequence (Fig. ~~1A~~ 2A) (24). This protein bound much more weakly to the cytoplasmic tail of MOR (Fig. ~~1A~~ 2A) and R DOR (25), consistent with a potential function in modulating DOR trafficking. We named this protein GASP for candidate G protein-coupled receptor-associated sorting protein. Psi-BLAST searches conducted with the GenBank database indicated that GASP is a previously unknown protein with human, rat, and murine homologs (26).

Please amend the paragraph [0245] at page 70 of the specification as shown below:

A rabbit polyclonal antibody was raised against the carboxyl-terminal 15 residue of GASP 1 (27). This antibody recognized a major immunoreactive protein in immunoblots of (untransfected) HEK 293 cell lysates, which co-electrophoresed with recombinant HA-tagged GASP 1 protein expressed in HEK 293 cells (Fig. 2B) and also has indistinguishable eletrophoretic mobility from recombinant GASP 1 produced by in vitro translation (Fig. ~~1A~~ 2A) (28, 29).

Please amend the paragraph [0247] at page 70 of the specification as shown below:

GST affinity chromatography was used to identify a COOH-terminal fragment of GASP (cGASP, corresponding to the COOH-terminal 497 residues of GASP, SEQ ID NO:8) that bound specifically to the DOR tail, consistent with the finding that several two-hybrid hits contained this portion of GASP (Fig. 3A). cGASP bound to the DOR tail with an apparent affinity comparable to that of full-length GASP (Fig. 3B) (24), as indicated by the similar fraction of GASP and cGASP recovered on beads when applied at similar concentrations and assayed by parallel GST binding (Figs. 2A and 3B). A GFP-tagged version of cGASP stably overexpressed in HEK293 cells coimmunoprecipitated with wild-type DOR but not MOR (Fig. 3C) (30), demonstrating that the specificity of the cGASP-DOR interaction observed in intact cells parallels that of the DOR-GASP interaction observed in vitro. cGASP was able to compete for binding of full-length GASP to the DOR tail in vitro (Fig. 3D) (31). Furthermore, GFP-cGASP, when highly overexpressed (Fig. 3E), markedly reduced the amount of endogenous GASP recovered in DOR immunoprecipitates (Fig. 3E),

suggesting that cGASP can function in intact cells as a dominant inhibitor of the interaction between endogenous GASP and DOR.